WEST Search History

DATE: Wednesday, October 29, 2003

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR		
L8	L7 and l3	5	L8
L7	(HSV or HSV-1 or herpes adj simplex) and (abrasion or scratch)	962	L7
L6	L5 and l3	20	L6
L5	(HSV or HSV-1 or herpes adj simplex) and (abra$5 or scratch)	1959	L5
L4	(HSV or HSV-1 or herpes adj simplex) and abra$5	1435	L4
L3	(HSV or HSV-1 or herpes adj simplex) with reactivation	117	L3
L2	(HSV or HSV-1 or herpes adj simplex) with reactivation and abrasion same (inoculat$7 or administer$8 or contact$3 or infect$5)	2	L2
L1	(HSV or HSV-1 or herpes adj simplex) with reactivation and abrasion same (inoculat$7 or administer$8 or contact$3)	2	L1

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Search PubMed for Herpes AND infection AND abrasion Preview Go

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#13	Search **Herpes AND infection AND abrasion**	11:55:25	8
#19	Search **Herpes AND reactivation AND inhibit** Field: **Title/Abstract,** Limits: **Publication Date to 2000/03/03**	11:53:06	22
#7	Search **Herpes AND reactivation AND model** Field: **Title/Abstract,** Limits: **Publication Date to 2000/03/03**	09:49:11	141
#16	Search **#13 AND #4**	09:39:36	0
#15	Search **#13 AND #6**	09:39:29	0
#14	Search **#13 AND #7**	09:39:13	0
#12	Search **Herpes AND infection and abrasion**	09:38:37	8
#9	Search **Herpes AND reactivation AND surface** Field: **Title/Abstract,** Limits: **Publication Date to 2000/03/03**	09:37:10	14
#8	Search **Herpes AND reactivation AND abrasion** Field: **Title/Abstract,** Limits: **Publication Date to 2000/03/03**	09:37:04	0
#6	Search **Herpes AND reactivation** Field: **Title/Abstract,** Limits: **Publication Date to 2000/03/03**	09:36:22	902
#5	Search **Herpes AND reactivation** Field: **Title/Abstract**	09:36:00	1163
#4	Search **Herpes AND reactivation**	09:35:49	1280
#3	Search **Herpes AND reactivation AND abrasion**	09:35:35	0
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#1	Search **HSV AND reactivation AND abrasion**	09:35:19	0

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Anti-interleukin-6 antibodies inhibit herpes simplex virus reactivation.

Kriesel JD, Gebhardt BM, Hill JM, Maulden SA, Hwang IP, Clinch TE, Cao X, Spruance SL, Araneo BA.

Department of Medicine, University of Utah School of Medicine, Salt Lake City 84132, USA.

Herpes simplex viruses (HSVs) infect epithelial cells, become localized in neurons, and can reactivate in response to a variety of stimuli, including ultraviolet light and hyperthermia. The sequence of gene activation during viral replication is known, but the molecular linkage between exogenous stimuli and HSV reactivation has not been determined. It was hypothesized that interleukin (IL)-6 acts as a signal between exogenous stimuli and neurons, stimulating HSV reactivation from latency. Mouse corneas were infected with HSV-1, and ocular reactivation was induced 5-7 weeks later by thermal stress or corneal exposure to ultraviolet light. Anti-IL-6 monoclonal antibodies were administered to the latently infected mice 8-12 h before the reactivation stimulus. Treatment with anti-IL-6 antibodies resulted in significantly lower frequencies of ocular reactivation compared with those in mice treated with a control immunoglobulin. These results support the hypothesis that IL-6 plays a role in HSV reactivation from latency.

PMID: 9086136 [PubMed - indexed for MEDLINE]

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1: Virology. 1988 Nov;167(1):302-5. Related Articles, Links

Herpes simplex virus latent infection: reactivation and elimination of latency after neurectomy.

Tenser RB, Edris WA, Hay KA.

Department of Medicine (Neurology), Pennsylvania State University College of Medicine, Hershey 17033.

Section of the sciatic nerve during the period of herpes simplex virus (HSV) latent infection was performed to evaluate residual latency in mouse dorsal root ganglion. In control mice without sciatic neurectomy, latency was present in 90-100%, while in those which underwent a neurectomy procedure, latent infection was surprisingly decreased to 28-50%. To investigate the hypothesis that the decrease of latency resulted from HSV reactivation and replication (with subsequent neuron destruction), groups of mice were treated with acyclovir to inhibit HSV reactivation, after having undergone a neurectomy procedure. Acyclovir treatment largely prevented the neurectomy-related elimination of latency and supported the hypothesized mechanism.

PMID: 2847420 [PubMed - indexed for MEDLINE]

Herpes virus infection of endothelium: new insights into atherosclerosis.

Jacob HS, Visser M, Key NS, Goodman JL, Moldow CF, Vercellotti GM.

Department of Medicine, University of Minnesota Medical School, Minneapolis.

Several pieces of evidence suggest that vascular endothelium may be a site of latent herpetic viral infection, and that activation of such infection might cause or aggravate atherosclerosis. The present studies which utilized HSV-1 infection of cultured endothelial monolayers, provide insights into two phenomena seemingly relevant in considerations of atherosclerosis. Thus, mechanisms are reported by which infected endothelium may be damaged by marginated inflammatory cells, and be transformed from an anticoagulant to a procoagulant tissue. First, granulocytes are attracted to, and avidly bind, endothelium infected for very brief periods. This interaction is associated with denudation of intact cells as well as actual cytolysis through release of PMN proteases and toxic oxygen species. Second, several potentially additive abnormalities of HSV-infected endothelium would seem to foster coagulation. These include: a) its loss of surface heparans and thrombomodulin; b) its inability to synthesize prostacyclin with associated incapacity to deter platelet adhesion; c) its disordered membrane lipid conformation which is likely associated with excessive surface thrombin generation; and d) its unique ability to generate and release tissue factor. We speculate that mechanical abrasion may reactivate latent herpes (HSV or CMV) infection in endothelial cells particularly those exposed to high shear forces--for instance, at vessel bifurcations. This may underlie the endothelial damage, clotting and atheroma formation commonly found at these sites.

Publication Types:
- Review
- Review, Tutorial

PMID: 1329303 [PubMed - indexed for MEDLINE]

9-(4-Hydroxybutyl)-N2-phenylguanine (HBPG), a thymidine kinase inhibitor, suppresses herpes virus reactivation in mice.

Gebhardt BM, Wright GE, Xu H, Focher F, Spadari S, Kaufman HE.

Lions Eye Research Laboratories, LSU Eye Center, Louisiana State University Medical Center School of Medicine, New Orleans 70112, USA.

In cells of the nervous system, which have little or no cellular thymidine kinase, the pharmacologic inhibition of viral thymidine kinase may prevent the reactivation of herpes virus, which requires phosphorylated thymidine for replication. We tested a newly synthesized inhibitor of viral thymidine kinase, 9-(4-hydroxybutyl)-N2-phenylguanine (HBPG) for its capacity to suppress the reactivation of herpes simplex virus type 1 (HSV-1) in vivo. Mice, latently infected with McKrae strain HSV-1, were treated with intraperitoneal injections of HBPG in a corn oil vehicle (200 mg/kg every 3 h for a total of ten doses), and subjected to hyperthermic stress to stimulate viral reactivation immediately before the third treatment. Three h after the last treatment, the mice were sacrificed, and the presence of infectious virus was determined by culture of ocular surface swabs and trigeminal ganglionic homogenates. Additionally, viral DNA in ganglionic extracts was analyzed by quantitative PCR. Controls included latently infected, stressed animals receiving injections of corn oil vehicle only, and latently infected, drug- and vehicle-treated, unstressed animals. HBPG had a statistically significant inhibitory effect on hyperthermia-induced viral reactivation. Homogenates of trigeminal ganglia and ocular surface swabs from HBPG-treated animals were less likely to contain infectious virus than those of infected, vehicle-treated, stressed controls ($P < 0.005$, ANOVA). Unstressed controls showed no reactivation. Quantitation of viral DNA in ganglionic extracts demonstrated a 100-fold reduction in the amount of viral DNA in the ganglia of HBPG-treated animals, compared with vehicle-treated controls ($P < 0.05$, ANOVA). The results indicate that HBPG has an inhibitory effect when given systemically for the suppression of herpes virus reactivation in mice.

PMID: 8783801 [PubMed - indexed for MEDLINE]